Clinical Investigation

Anaesthesia for awake craniotomy: A retrospective study of 54 cases

Address for correspondence:
Dr. Girija Prasad Rath,
Department of Neuroanaesthesiology,
Neurosciences Centre,
All India Institute of Medical Sciences,
New Delhi - 110 029, India.
E-mail: girijarath@yahoo.co.in

Navdeep Sokhal, Girija Prasad Rath, Arvind Chaturvedi, Hari Hara Dash, Parmod Kumar Bithal, P Sarat Chandra[1]

Departments of Neuroanaesthesiology and [1]Neurosurgery, All India Institute of Medical Sciences, New Delhi, India

Access this article online

Website: www.ijaweb.org

DOI: 10.4103/0019-5049.156878

Quick response code

ABSTRACT

Background and Aims: The anaesthetic challenge of awake craniotomy is to maintain adequate sedation, analgesia, respiratory and haemodynamic stability in an awake patient who should be able to co-operate during intraoperative neurological assessment. The current literature, sharing the experience on awake craniotomy, in Indian context, is minimal. Hence, we carried out a retrospective study with the aim to review and analyse the anaesthetic management and perioperative complications in patients undergoing awake craniotomy, at our centre. **Methods:** Medical records of 54 patients who underwent awake craniotomy for intracranial lesions over a period of 10 years were reviewed, retrospectively. Data regarding anaesthetic management, intraoperative complications and post-operative course were recorded. **Results:** Propofol (81.5%) and dexmedetomidine (18.5%) were the main agents used for providing conscious sedation to facilitate awake craniotomy. Hypertension (16.7%) was the most commonly encountered complication during intraoperative period, followed by seizures (9.3%), desaturation (7.4%), tight brain (7.4%), and shivering (5.6%). The procedure had to be converted to general anaesthesia in one of patients owing to refractory brain bulge. The incidence of respiratory and haemodynamic complications were comparable in the both groups ($P > 0.05$). There was less incidence of intraoperative seizures in patients who received propofol ($P = 0.03$). In post-operative period, 20% of patients developed new motor deficit. Mean intensive care unit stay was 2.8 ± 1.9 day (1–14 days) and mean hospital stay was 7.0 ± 5.0 day (3–30 days). **Conclusions:** 'Conscious sedation' was the technique of choice for awake craniotomy, at our institute. Fentanyl, propofol, and dexmedetomidine were the main agents used for this purpose. Patients receiving propofol had less incidence of intraoperative seizure. Appropriate selection of patients, understanding the procedure of surgery, and judicious use of sedatives or anaesthetic agents are key to the success for awake craniotomy as a procedure.

Key words: Anaesthesia, awake craniotomy, complication, conscious sedation, regional scalp block

INTRODUCTION

Awake craniotomy is performed for localization and resection of epileptic focus or for resection of tumours located near the eloquent areas of brain such as Broca's speech area, Wernicke's speech area or motor strip region.[1] It allows intraoperative neurological testing which facilitates optimal tumour resection and minimizes post-operative neurological dysfunction. However, this technique brings challenges both to the neurosurgeon and anaesthesiologist. The goal of anaesthesia is not only to maintain adequate sedation, analgesia, respiratory and haemodynamic stability but also to provide an awake yet comfortable and co-operative patient for proper neurological testing. There is a growing trend towards preference of awake craniotomy as a procedure, over the last few decades, and similar pattern has been observed at our institution.

How to cite this article: Sokhal N, Rath GP, Chaturvedi A, Dash HH, Bithal PK, Chandra PS. Anaesthesia for awake craniotomy: A retrospective study of 54 cases. Indian J Anaesth 2015;59:300-5.

During this procedure, there is always a concern about patient acceptability, cooperation and safety.[2] Proper selection of patient, counselling, adequate measures to make the patient comfortable during the surgery and good communication with the patient are essential components of such procedures.[3] Despite all the above measures various adverse events have been reported during the perioperative period which require intervention. Sometimes, the procedure needs to be converted to general anaesthesia (GA) either due to non-cooperation or agitation of the patient.[4] Nevertheless, current literature sharing the experience on awake craniotomy, in Indian context, is rare. Hence, we planned to review and analyse the anaesthetic management and perioperative complications in patients undergoing awake craniotomy at our centre.

METHODS

After clearance from Institution Ethics Committee, medical records of patients who underwent awake craniotomy at our hospital during January 2001 to December 2010 were retrospectively reviewed. Data pertaining to pre-anaesthetic evaluation, intraoperative management, and post-operative course were collected. The pre-operative data included age, sex, weight, American Society of Anesthesiologists (ASA) physical status, relevant history, existing neurological deficits, associated co-morbidities, and pre-operative investigations performed. Airway status with Mallampati (MP) grade was also noted. Intraoperative data included anaesthetic technique, duration of surgery, and intraoperative complications encountered such as bradycardia, tachycardia, hypotension, and hypertension (20% changes in baseline values), pain, hypoxia ($SpO_2 \leq 90\%$), tight brain, seizure, cough, and any other complications. Post-operative data included occurrence of nausea and vomiting, seizures, fever, surgical and neurological complications, progression or occurrence of new deficits, histopathological character of lesion, and duration of Intensive Care Unit and hospital stay.

The patients were evaluated during the pre-operative visit by the concerned neuroanaesthesiologist and the procedure was explained in detail. They were instructed to continue their ongoing anti-epileptic and anti-hypertensive medications along with corticosteroids and were kept fasting from mid-night. Obese patients with history of obstructive sleep apnoea and those with anticipated difficult airway were not considered as candidates for this procedure.

In the operating room, oxygen mask or nasal cannula for oxygenation with end-tidal carbon-dioxide monitoring was attached. Standard monitors such as electrocardiogram, pulse oximeter and non-invasive blood pressure were connected. Urinary catheter was inserted to monitor the urine output. Invasive blood pressure was monitored after arterial cannulation with local anaesthetic (LA) infiltration in radial or dorsalis pedis artery. Each patient received regional scalp block with 0.25–0.5% bupivacaine. Supra-orbital, supra-trochlear, lesser and greater occipital, zygomatico-temporal and auriculo-temporal nerves were blocked with 3–5 ml of LA solution at each site. Prior to block, fentanyl, bolus dose of propofol or dexmedetomidine were administered, IV. All patients received LA infiltration at incision site. Bispectral index (BIS®) monitoring (A-2000; Aspect Medical Systems, Newton, MA, USA) was done to titrate the requirement of sedatives and hypnotics, whenever available. Monitored anaesthesia care (MAC)/conscious sedation was used for all patients as the anaesthetic technique. Intermittent bolus or continuous intravenous (IV) infusion of fentanyl (0.25–1.5 μg/kg/h) and propofol (25–200 μg/kg/min) was used with titration to the patient's haemodynamics and procedural needs. After the availability of dexmedetomidine in our hospital since 2010, it was used for conscious sedation either as the sole agent or in combination with titrated doses of fentanyl. It has been used as an IV bolus of 1 μg/kg over 10 min followed by 0.2–0.7 μg/kg/h for maintenance of sedation.

STATA 11.0 (College Station, Texas, USA) was used for statistical analysis. Demographic data are described in mean ± standard deviation and range format. Values are mentioned in number and percentage. Fisher's exact test was used for sub group analysis to detect non-random association between categorical variables.

RESULTS

The study sample included 54 patients who underwent awake craniotomy for intracranial lesions (tumour and epileptic foci) over a period of 10 years. All patients belonged to ASA physical status I/II [Table 1]. MP grade III/IV airway was observed in 6 (11.1%) patients. Two patients underwent the procedure for recurrent tumours. The procedure was converted to GA in one patient (1.9%) after intraoperative brain bulge. One patient required post-operative non-invasive positive pressure ventilation after intraoperative venous air embolism (VAE) and subsequent respiratory distress.

The most common pre-operative symptoms were seizures (89%) followed by headache (35%), limb weakness (35%), dysphasia (17%), facial nerve paresis (17%), diminished vision (11%), memory loss (7%), parietal lobe symptoms such as dyscalculia and hemineglect (4%), and paraesthesia (2%). The symptoms were present either alone or in combination.

Hypertension was the most common pre-operative medical condition present in 14.8% of patients. Most of the lesions were located in left side of brain (67%). Tumours were localized around different areas; majority of which were in the frontal lobe (61.1%). Sedation was monitored with BIS or entropy in 26% of patients, the monitoring value was targeted at 60–80.

Propofol and fentanyl combination was the most commonly used anaesthetic regimen to provide MAC in 44 (81.5%) patients (propofol-fentanyl group). Dexmedetomidine was used for conscious sedation in 10 (18.5%) cases (dexmedetomidine group). In six patients (11%) it was used as the sole agent, in three patients it was used along with fentanyl whereas one patient required fentanyl and propofol [Table 2]. The episodes of airway obstruction leading to desaturation, and hypertension were more in propofol group as compared to dexmedetomidine [Table 2]. However, it was statistically not significant ($P > 0.05$).

Hypertension (16.7%) was the most commonly encountered complication during intraoperative period which was followed by seizures (9.3%), desaturation and hypoxaemia (7.4%), tight brain (7.4%), shivering (5.6%), apnoea (3.7%), pain (3.7%) and cough (3.7%). Hypotension, VAE, and snoring were observed, each in one patient [Figure 1].

During intraoperative period, the limb paresis was exacerbated in 7 (13%) patients whereas 4 (7%) patients developed new deficits. In post-operative period, 11 (20%) patients developed new deficit, while paresis was exacerbated in 8 (15%) patients. In two patients, new-onset paresis developed in post-operative period which was transient. Dysphasia exacerbated in one patient intraoperatively and another patient, post-operatively. In the intraoperative period, three patient (6%) developed new-onset dysphasia, they continued to be dysphasic in post-operative period.

Five (9.3%) patients developed intraoperative seizure which was continued in the post-operative period in two patients. Five more patients developed seizures in the post-operative period making overall seven (13%) patients with post-operative seizures. All these patients suffered from seizures during pre-operative period. In fentanyl-propofol group, two patients developed

Table 1: Demographic data (*n*=54)

Parameters	Values Mean±SD (range)/number (%)
Age (year)	36.1±14.3 (17-72)
Sex	
Male	43 (79.6)
Female	11 (20.4)
Weight (kg)	61.4±11.2 (45-98)
ASA physical status	
1	41 (75.9)
2	13 (24.1)
Laterality of lesions	
Right	18 (33)
Left	36 (67)
Recurrent tumour	2 (3.7)
Duration of surgery (min)	268±45.7 (165-390)
ICU stay (days)	2.8±1.9 (1-14)
Hospital stay (days)	7.0±5.0 (3-30)

ASA – American Society of Anesthesiologists; ICU – Intensive Care Unit; SD – Standard deviation

Table 2: Intraoperative complications (*n*=54)

Complication	Fentanyl-propofol *n*=44	Dexmedetomidine *n*=10	*P*
Tachycardia	0	1	0.18
Hypertension	9	0	0.18
Hypotension	0	1	0.18
Desaturation	3	1	0.57
Apnoea	2	0	1
Tense brain	4	0	1
Pain	2	0	1
Movement	2	0	1
Seizure	2	3	0.03*
Shivering	3	0	1
Cough	1	1	0.33
VAE	0	1	0.18
Snoring	1	0	1

**P*<0.05=significant. VAE – Venous air embolism

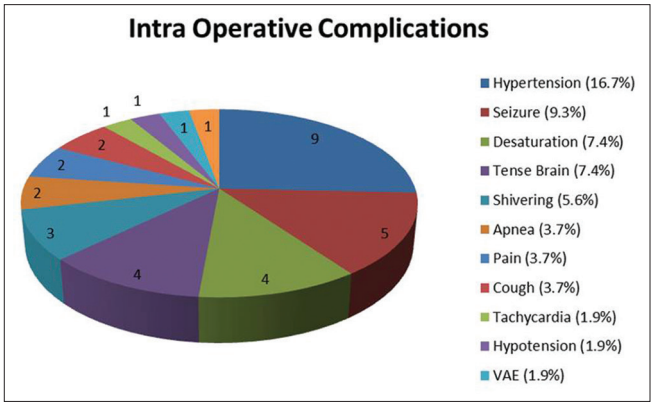

Figure 1: Intraoperative complications. VAE – Venous air embolism

intraoperative seizures while in dexmedetomidine group three patients had seizures ($P = 0.03$).

DISCUSSION

Awake craniotomy is carried out mostly for epilepsy surgery and excision of brain tumours located near eloquent areas of brain as it allows for cortical mapping and avoids complications inherent to GA.[5] Anaesthesia techniques for awake craniotomy include MAC or Conscious Sedation and Asleep Awake Asleep technique (AAA). In AAA, the airway is secured with laryngeal mask airway (LMA) or endotracheal tube (ETT), and the patients are kept on spontaneous or mechanical ventilation. LMA /ETT is removed and the patient is allowed to wake up for neurological testing. In MAC, the drug may be administered by intermittent bolus, continuous infusion, target-controlled infusion, patient-controlled analgesia or a combination and the patients maintain their airway and take spontaneous breaths. The commonly used anaesthesia regimen for conscious sedation include infusion of propofol with short-acting opioids such as fentanyl or remifentanil.[6,7] In our study, this propofol-based sedation technique was utilized in 44 out of 54 patients (81.5%). Propofol is a short-acting sedative with easy titratability. It has antiemetic and anticonvulsant effects. It decreases cerebral oxygen consumption and reduces intracranial pressure, and does not interfere with electrocorticography (ECoG) recording if infusion is stopped 15 min before brain mapping.[8,9] Airway compromise is uncommon in AAA technique but the incidences of hypertension, hypotension, and tachycardia were significantly more as compared to endotracheal anaesthesia technique.[10]

Blanshard *et al*.[11] reported anaesthetic management and complications of neuroleptanaesthesia in 241 patients operated awake for intracranial tumours. One patient (0.4%) required conversion to GA because of generalised convulsion with loss of airway patency. Similar complication was observed in one of our patient where the procedure was converted to GA after intraoperative severe brain bulge which was refractory to treatment with mannitol and furosemide.

Another patient, in this series, developed VAE and presented with intraoperative cough followed by tachycardia and desaturation. The patient was managed with oxygen via mask; however, during post-operative period non-invasive ventilation was required. Cough during awake craniotomy should raise the high suspicion for VAE. Balki *et al*.[12] reported VAE in one patient who developed similar symptoms.

Dexmedetomidine is a highly selective α_2-agonist which offers unique advantages of sedation, anxiolysis and analgesia without respiratory depression. It has minimal interference with ECoG in dose range of 0.2–0.5 μg/kg/hr.[13,14] In our study, dexmedetomidine was used with and without fentanyl in 10 cases with complication rate comparable to fentanyl-propofol group. Ard *et al*.[15] reported a case series of 17 patients undergoing awake craniotomy with dexmedetomidine infusion and observed no major complication. In our series, an episode of hypotension was encountered in a patient sedated with dexmedetomidine after bolus dose of fentanyl was given which was managed with IV fluids and mephentermine. The most common complication in this study was hypertension (17%) which was comparable to the incidences reported by the literature.[15,16]

Seizure was another major complication observed during awake craniotomy, mostly during cortical stimulation. Irrigation with cold saline has been suggested for control of intraoperative seizures.[17] Aggressive IV anti-epileptic medication with propofol, midazolam or thiopentone helps further with a secured airway if, seizure becomes generalised. The incidence of seizures during intraoperative period has been reported to be 3–10%. Serletis and Bernstein[5] and Sinha *et al*.[16] reported the incidences of seizures as 9.5% and 5%, respectively which similar to our study. The lesser incidence of intraoperative seizure in propofol-fentanyl group might be due to anti-epileptic activities of propofol.[18] Sinha *et al*.[16] used double doses of anti-epileptics pre-operatively, and midazolam intraoperatively for control of seizures. In our hospital, cold saline irrigation, infusion of phenytoin, and IV propofol are being used for this purpose.

Airway obstruction remains a major problem during awake craniotomy. Four of our patients (7.4%) were complicated by desaturation but they responded well to dose reduction and O_2 supplementation. Manninen *et al*.[6] reported an overall 16% incidence of airway obstruction, where five patients responded to jaw lift, one required nasal airway whereas another one required mask ventilation. Four percent of our patients developed apnoea; they responded well to manual stimulation and O_2 supplementation by mask. One such episode occurred after propofol bolus given to prevent movement of the patient. Continuous positive airway

pressure and LMA after propofol have been used in an apnoeic patient, to support ventilation.[16] Fibreoptic bronchoscope-guided intubation has been suggested following desaturation.[10] In patients having fixed head and restricted access to airway, the role of LMA, intubating LMA, combined oro-pharyngeal airway, and fibreoptic bronchoscope-guided intubation have been proposed to overcome inadequacy in ventilation. NIPPV in the form of biphasic positive airway pressure or proportional assisted ventilation has been employed for airway management during awake craniotomy.[19]

None of our patients developed intraoperative nausea and vomiting. It was possibly because all patients received antiemetic premedication. Moreover, use of steroids as anti-oedema measures and propofol for sedation might have helped further to prevents such episodes. The incidence of nausea and vomiting has been reported to be as high as 70% in some studies.[20] This high incidence may be explained by non-inclusion of propofol in the anaesthetic protocol.

Intraoperative tight brain was found in 7% of patients, in our series. During these episodes, the patients were encouraged to self-hyperventilate, and received mannitol and furosemide. However, during one such episode the procedure was converted to GA owing to refractory brain bulge. Sinha *et al*.[16] observed tight brain in 14% of patients. The lesser incidence of brain bulge in our patients may be explained by prophylactic administration of mannitol at the time of skin incision.

Intraoperative shivering may be problematic for patient being operated awake as it increases oxygen consumption, masquerades focal seizures and disturbs surgical procedure. Although the operating room temperature was kept at 22–25°C, shivering was noticed in 5.6% patients and pethidine or tramadol was administered to control shivering, despite the intraoperative use of forced-air warming system.

In 14 of our patients, BIS® or Entropy® were used to titrate the level of sedation. Use of BIS® is reported to reduce respiratory depression.[16] However, in this study, no difference was observed in intraoperative complication rate whether these monitoring modalities were used or not. Nevertheless, it has been maintained that BIS values may not correlate correctly with patient consciousness, in patients on anti-epileptic medication.[21]

Despite the use of ECoG and cortical stimulation, 31% of our patients developed post-operative motor deficits and 9% developed speech deficits or dysphasia. Post-operative paresis increased in 8 (15%) patient and new defect developed in 11 (20%) patients; in two (3.7%) patients it was transient. Blanshard *et al*.[11] found 11% transient deficit and 6% permanent deficits. Sinha *et al*.[16] found transient deficit of motor function in 24% patients whereas none of their patients suffered permanent deficit. Higher incidence of neurologic deficit observed in this study may be explained by the steep learning curve of our neurosurgeons.

Blanshard *et al*.[11] advocated proper patient selection for early discharge as 8% of patients were discharged within 6 h and 31% on the next day. The mean hospital stay of our patient population was 7.0 ± 5.0 day with a maximum stay of 30 days. One patient was complicated by post-operative haematoma and residual tumour oedema underwent decompressive craniotomy followed by tracheostomy. This patient was successfully decannulated later and discharged without neurological deficit. The reported incidence of post-operative haematoma in other studies is 1.1–3%[5,11] which is similar to our study.

CONCLUSION

'Conscious sedation' was the technique of choice for awake craniotomy at our institution. Propofol, fentanyl or dexmedetomidine were the main agents used for this purpose. Patients receiving propofol had fewer incidences of intraoperative seizures. The incidence of neurological deficits was more owing to steep learning curve of our neurosurgeons. Proper selection of patient, understanding of the surgical procedure, and judicious use of sedatives or anaesthetic agents are of paramount importance to carry out this procedure smoothly.

REFERENCES

1. Sahjpaul RL. Awake craniotomy: Controversies, indications and techniques in the surgical treatment of temporal lobe epilepsy. Can J Neurol Sci 2000;27 Suppl 1:S55-63.
2. Danks RA, Rogers M, Aglio LS, Gugino LD, Black PM. Patient tolerance of craniotomy performed with the patient under local anesthesia and monitored conscious sedation. Neurosurgery 1998;42:28-34.
3. Whittle IR, Midgley S, Georges H, Pringle AM, Taylor R. Patient perceptions of "awake" brain tumour surgery. Acta Neurochir (Wien) 2005;147:275-7.
4. Piccioni F, Fanzio M. Management of anesthesia in awake craniotomy. Minerva Anestesiol 2008;74:393-408.
5. Serletis D, Bernstein M. Prospective study of awake craniotomy used routinely and nonselectively for supratentorial tumors. J Neurosurg 2007;107:1-6.
6. Manninen PH, Balki M, Lukitto K, Bernstein M. Patient satisfaction with awake craniotomy for tumor surgery: A

comparison of remifentanil and fentanyl in conjunction with propofol. Anesth Analg 2006;102:237-42.
7. Khalifah N, Herrick I, Megyesi J, Parrent A, Steven D, Craen R. Patient satisfaction following awake craniotomy. Saudi J Anaesth 2008;2:52-6.
8. Soriano SG, Eldredge EA, Wang FK, Kull L, Madsen JR, Black PM, *et al.* The effect of propofol on intraoperative electrocorticography and cortical stimulation during awake craniotomies in children. Paediatr Anaesth 2000;10:29-34.
9. Herrick IA, Craen RA, Gelb AW, McLachlan RS, Girvin JP, Parrent AG, *et al.* Propofol sedation during awake craniotomy for seizures: Electrocorticographic and epileptogenic effects. Anesth Analg 1997;84:1280-4.
10. Skucas AP, Artru AA. Anesthetic complications of awake craniotomies for epilepsy surgery. Anesth Analg 2006;102:882-7.
11. Blanshard HJ, Chung F, Manninen PH, Taylor MD, Bernstein M. Awake craniotomy for removal of intracranial tumor: Considerations for early discharge. Anesth Analg 2001;92:89-94.
12. Balki M, Manninen PH, McGuire GP, El-Beheiry H, Bernstein M. Venous air embolism during awake craniotomy in a supine patient. Can J Anaesth 2003;50:835-8.
13. Bekker A, Sturaitis MK. Dexmedetomidine for neurological surgery. Neurosurgery 2005;57:1-10.
14. Souter MJ, Rozet I, Ojemann JG, Souter KJ, Holmes MD, Lee L, *et al.* Dexmedetomidine sedation during awake craniotomy for seizure resection: Effects on electrocorticography. J Neurosurg Anesthesiol 2007;19:38-44.
15. Ard JL Jr, Bekker AY, Doyle WK. Dexmedetomidine in awake craniotomy: A technical note. Surg Neurol 2005;63:114-6.
16. Sinha PK, Koshy T, Gayatri P, Smitha V, Abraham M, Rathod RC. Anesthesia for awake craniotomy: A retrospective study. Neurol India 2007;55:376-81.
17. Sato K, Kato M. Intraoperative neurological monitoring in awake craniotomy. J Anesth 2008;22:493-7.
18. Herrick IA, Craen RA, Gelb AW, Miller LA, Kubu CS, Girvin JP, *et al.* Propofol sedation during awake craniotomy for seizures: Patient-controlled administration versus neurolept analgesia. Anesth Analg 1997;84:1285-91.
19. Yamamoto F, Kato R, Sato J, Nishino T. Anaesthesia for awake craniotomy with non-invasive positive pressure ventilation. Br J Anaesth 2003;90:382-5.
20. Gignac E, Manninen PH, Gelb AW. Comparison of fentanyl, sufentanil and alfentanil during awake craniotomy for epilepsy. Can J Anaesth 1993;40:421-4.
21. Pemberton PL, Dinsmore J. Bispectral index monitoring during awake craniotomy surgery. Anaesthesia 2002;57:1244-5.

Source of Support: Nil, **Conflict of Interest:** None declared

Announcement

CALENDAR OF EVENTS OF ISA - 2015

Certain important dates are given here for the members. All the applications should be sent by registered post (with Acknowledgement Due)

Date	Name of the Award/Post	Application has to be sent to
30th June 2015	Bhopal Award for Academic Excellence	Hony. Secretary, ISA
15th August 2015	Prof. A. P. Singhal Life Time Achievement Award	Hony. Secretary, ISA
31st October 2015	Dr. (Mrs.) Rukmini Pandit Award - Publication format along with Conference Presentation Certificate	Hony. Secretary, ISA
31st October 2015	Y. G. Bhoj Raj Award - Best Review Article in IJA	Hony. Secretary, ISA
31st October 2015	Dr. Kop's Award	Chairman Scientific committee of ISACON with a copy to Hony Secretary ISA
27th November 2015	Dr. TN Jha Memorial & Dr. KP Chansoriya Travel grant	Hony. Secretary, ISA
27th November 2015	Late Dr. Venkata Rao Memorial Oration	Hony. Secretary, ISA
27th November 2015	Ish Narani Best Poster Award	Chairman Scientific Committee ISACON
28th November 2015	ISA GOLDCON QUIZ Competition	Chairman Scientific Committee ISACON
28th November 2015	Awards for 1. Best City Branch 2. Best State Branch 3. Best Metro Branch 4. Public Awarness Individual 5. Public Awarness City 6. Public Awarness State 7. Ether Day State 8. Ether Day City 9. Membership Drive % (State) 10. Membership Drive No.s (State) 11. Individual Drive	Hony. Secretary, ISA

Dr. Venkatagiri K M
"ASHWATHI", Opp. Ayyappa Temple, Nullippady, Kasaragod - 671121, Kerala
Email: isanhq@gmail.com / secretaryisanhq@gmail.com/ isanhq@isaweb.in Mobile: 093880 30395